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SPONSORED PROJECT INITIATION

Date: 8/29/78

Project Title: Mechanical Properties and Function of Pericardium

Project No: E-23-638

Project Director: Dr. Raymond P. Vito

Sponsor: Georgia Heart Association, Inc.

Agreement Period: From 7/1/78 Until 6/30/79 (Grant Period)

Type Agreement: Grant -- Letter dated 7/13/78, w/check No. 937 (dtd. 7/10/78)

Amount: $4,400 (Partially funded at $2,200 through 12/31/78)

Reports Required: Terminal Report

Sponsor Contact Person (s):

Technical Matters

Contractual Matters (thru OCA)

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Defense Priority Rating: N/A

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CA-3 (3/76)

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Date: October 12, 1979

Project Title: Mechanical Properties and Function of Pericardium

Project No: E-23-638

Project Director: Dr. Raymond P. Vito

Sponsor: Georgia Heart Association, Inc.

Effective Termination Date: September 1, 1979

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CA-4 (1/79)

E-25-620

Mechanical Properties and Function of Pericardium

a report to the
Georgia Heart Association

by
Raymond P. Vito
Principal Investigator

Period of Support: 6/1/78 - 9/1/79
Date of Report : 9/17/79

1. Scientific Summary

1.1 Experimental Results

The mechanical properties of dog pericardium were studied in twenty uni-axial experiments. In each experiment, the stretching force and elongation in the direction of the force were measured. Results indicate that:

a) the resulting force versus elongation curves are relatively independent of the rate of loading.

b) the pericardium is viscoelastic: loading and unloading curves are not the same.

c) the response, in constant stretch rate experiments, approaches a steady state after five or six load/unload cycles.

These results are representative of soft tissue mechanical behavior (Fung 1973), though they had not been previously verified for the pericardium.

The experiments (to the author's knowledge) are the most complete study of pericardial mechanical properties yet conducted. Details may be found in Vito (1979).

1.2 Analytical Results

The complicated nonlinear viscoelastic behavior of the specimens complicates the analysis. The following assumptions were made:

a) individual load/unload cycles were analyzed assuming elastic (time independent) behavior.

b) the mechanical properties were assumed to be direction independent (isotropic behavior).

c) the tissue was assumed homogeneous, i.e., microstructure was ignored.

d) the tissue was assumed incompressible, i.e., there is no volume change associated with the deformation.

These assumptions are justified primarily by the preliminary nature of this study. Additional justifications may be found in Vito (1979).

For the stated assumptions, the mechanical response of the tissue is completely determined by an elastic potential function W. Uni-axial experiments do not uniquely determine the function W. However, any suitable function must agree with the known data. Several functional forms for W have been proposed in the literature on soft tissues, e.g. Vito (1973), Fung (1973). The experimental data was in good agreement, in a least squared sense, with a function containing two material parameters. This function is similar in form to that characterizing rubber elasticity.

The two material parameters and their distributions were determined. Results and additional details may be found in Vito (1979).

It should be noted that the determination of a constitutive law, which reduces, in the present case, to the determination of the function W, is fundamental to the consideration of the mechanical functions of the pericardium. Additional information concerning W can be obtained from bi-axial mechanical tests. Bi-axial tests are mechanical tests in which the specimens are stretched in each of two orthogonal directions. These experiments are in progress. Preliminary results indicate that the functional form for W mentioned above is suitable for matching the bi-axial data as well.

1.3 Model Results

Mechanical modelling of the heart may be useful in the interpretation of clinical measures of cardiac function as well as in understanding the basic physiology of heart muscle in health and disease. Many such models have been proposed in the literature but none, to the author's knowledge, have included the pericardium.

Accordingly, the effects of the pericardium on a simple, but representative, heart model were considered. The model makes use of the experimental data discussed above.

The effects of pericarditis (pericardial thickening) and effusion (increased pericardial fluid volume) were simulated (Vito 1979, Vito et al 1979). In both cases the pericardium had significant effects on the pericardial fluid pressure and on the distensibility of the heart. Similar effects on the stresses in the ventricular wall and the work load on the heart are expected.

The model is a simple one and open to criticism. It does appear, however, that the mechanical properties of the pericardium are of fundamental importance in the consideration of any cardiac model.

Publications

Two publications, Vito 1979 and Vito et al 1979, resulted from this work (copies attached).

Research Continuation

As previously mentioned, bi-axial experiments are in progress. A proposal seeking support for continuing this research has been prepared for submission to NSF.

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J. Biomechanics Vol. 12, pp. 587–592.
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0021-9290/79/0801-0587 $02.00/0

THE ROLE OF THE PERICARDIUM IN CARDIAC MECHANICS

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Abstract – This paper considers the effects of the pericardium on a mechanical model of the heart. The model, based on the theory of large elastic deformation, makes use of experimental data for the pericardium reported herein.

INTRODUCTION

The pericardium is the tough fibrous sac enclosing the ıeart of most vertebrates, including man. It serves to ıold the heart in a fixed position and to isolate and protect it from other thoracic structures. The pericardial sac contains fluid (pericardial fluid – similar to blood serum) which serves to lubricate the outer urfaces of the heart.

Anatomically, the pericardium consists of a thin nembrane covering the heart (the visceral pericarlium), surrounded by a thick fibrous sac (the parietal ericardium). The sac is generally attached to the great essels, the sternum, the vertebral column and the iaphragm (Holt, 1970).

Histologically, the parietal pericardium consists of ıterlaced collagen and elastin fibers, relative comosition being a function of age in man (Holt, 1970).

The pericardium is not essential to life and its emoval does not result in any obvious disability, ıough there may be long term effects such as cardiac ypertropy (Fowler, 1970). Its constrictive function, owever, can have fatal consequences in pathological tuations, such as cardiac tamponade (impairment of iastolic filling of the heart caused by an unchecked se in intrapericardial pressure) and constrictive periırditis (inflammation of the pericardium, resulting in onstriction of the heart).

From this discussion, it appears that the mechanical sponse of the pericardium can have important effects n cardiac function. Its restriction of free myocardial pansion can effect myocardial wall stresses. Elevated ress may be responsible for ischemia of the ventriılar wall (Mirshy, 1973). This may be important in ses of pericarditis associated with myocardial farction.

Attempts to model the heart (Demiray, 1976; lirsky, 1973; Voukydis, 1972a, b, c; Janz *et al.*, 1972; ould *et al.*, 1972; Ghista *et al.*, 1969; Mirsky, 1969; hista *et al.*, 1968; Wong *et al.*, 1968) have not cluded the pericardium. These models may be critized in many ways. Nevertheless, much current search in biomechanics has as its goal a model of cardiac function in health and disease. Such a model must include the effects of the pericardium.

Very little is known about the mechanical response of the pericardium. Only three papers (Rabkin *et al.*, 1974, 1975; Hildebrandt *et al.*, 1969) could be found from 1940. These papers are based on a small number of experiments, six in the case of Rabkin *et al.* (1974, 1975) and an unspecified number in the case of Hildebrandt *et al.* (1969).

In this paper, the one-dimensional elastic response of dog pericardium was determined in 20 experiments. The results were analyzed statistically and used to model the effect of the pericardium on the heart.

EXPERIMENT

A total of twenty experiments were conducted using tissue from 10 dogs of varying weight, age and sex. All experiments were conducted within 24 hr of sacrifice; the tissue being maintained in normal saline at 5°C prior to use. All experiments were conducted with the tissue immersed in normal saline at 37°C.

Specimens approximately 3.5 cm in length and 0.5 cm in width were prepared. Hildebrandt *et al.* (1969) observed an apparent insensitivity of results to orientation of the specimen. Hence the orientations of the specimens were not recorded.

The ends of the tissue were sandwiched between square pieces of balsa wood approximately 0.5 cm on a side. The balsa was then tied securely using 000 silk. Fish hooks, passing through the balsa and tissue, were used to attach the tissue to fine jewelers chain. The chain was in turn attached to a force transducer (Statham) and to the movable rod of a one-dimensional testing device.

Specimens were loaded to 49,000 dyn (50 g-force) and unloaded at a constant strain rate to zero force. Since strain rate had little effect on the resulting force deformation curves, a constant rate of 0.25 cm/min was used. Between 3 and 5 load–unload cycles were required to precondition the specimens (Fung, 1973).

Data consists of the undeformed dimensions and continuous measurements of force and change in length. Length change was measured using a DCDT

* *Received* 10 *October* 1978.

*Acknowledgement of the support of the Georgia Heart Association, though in the original manuscript, was omitted from the final proof. Unfortunately, I did not catch this error. The next Journal issue will contain an errata with the acknowledgement.

(Hewlett-Packard). Data was taken digitally using a microprocessor (Intel 8080) controlled twelve bit A/D converter (Burr Brown). Approximately 240 data sets were taken per load-unload cycle. All data was stored on a floppy disk (Digital Systems) and later transmitted, using a modem, to the Georgia Tech Computer Center for analysis.

ANALYSIS AND DATA REDUCTION

The specimens were assumed to behave as homogeneous, isotropic, incompressible materials (Hildebrandt *et al.*, 1969). Though the material responds viscoelastically, each load or unload cycle was modeled as if the material response were purely elastic. This kind of analysis is fundamental to the development of more complex nonlinear viscoelastic models (Fung, 1973).

The elastic response is completely determined by the response function $W(I_1, I_2)$ (Green and Zerna, 1954), where I_1 and I_2 are the strain invariants. In particular, the one-dimensional response is easily shown to be (Green and Adkins, 1960)

$$T(\Lambda) = \left(\Lambda - \frac{1}{\Lambda^2}\right)\Phi + \left(1 - \frac{1}{\Lambda^3}\right)\Psi, \qquad (1)$$

where

$$\Phi = 2\frac{\partial W}{\partial I_1} \qquad \Psi = 2\frac{\partial W}{\partial I_2}$$
$$I_1 = \Lambda^2 + \frac{2}{\Lambda} \qquad I_2 = 2\Lambda + \frac{1}{\Lambda^2}. \qquad (2)$$

T is the stress referenced to the undeformed cross sectional area and Λ is the extension ratio.

One-dimensional data is not sufficient for the unique determination of W. Such a determination requires a bi-axial test in which the principal strains may be independently varied. Nevertheless, any suitably chosen form for W must agree with all known results.

Several suitable forms for W have been proposed in the literature. In particular, we note the exponential-polynomial form of Fung (1973), the exponential form of Demiray and Vito (1976) and the polynomial form of Vaishnav *et al.* (1972).

The experimental data reported here was found to be in good agreement, in a least acquired sense, with predictions of the following two parameter model

$$W(I_1, I_2) = \alpha(I_1 - 3)^{\beta}. \qquad (3)$$

This is a modification of the well known Mooney model for rubber and is in this way similar to that used by Hildebrandt *et al.* (1969) in their analysis of uniform bi-axial tests of pericardium.

The constants α and β were determined for a least squared fit to the data, using a simple algorithm found in Berghaus (1977).

RESULTS

In three experiments, the tissue was observed to slip in the supports and hence these data were not used.

Experimental data and theoretical predictions for a representative load cycle are shown in Fig. 1. Figures 2 and 3 give the distribution of results for 60 load and unload cycles. Considerable scatter, typical of biological experiments, is evident. A summary of results is given in Table 1.

MODEL

In this section, the above results will be used to illustrate the effects of the pericardium on a model for heart. The model, based on the theory of large elastic deformation, is similar to that given by Mirsky (1973) and Demiray (1976), in that the ventricle is considered to be a sphere composed of a homogeneous, isotropic, incompressible, elastic material. The pericardium, not previously considered, will be assumed to be a second sphere similarly composed and concentric with the first.

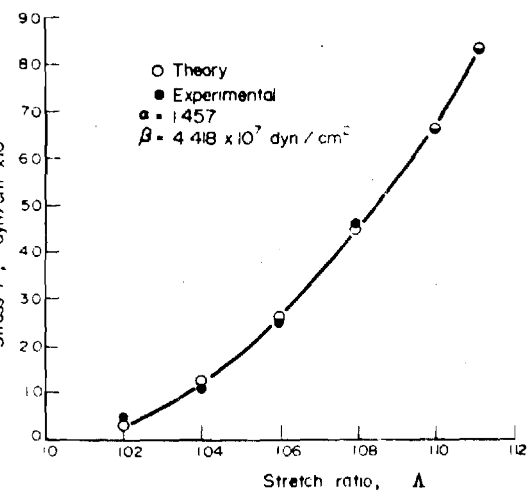

Fig. 1. Representative plot of stress vs stretch ratio.

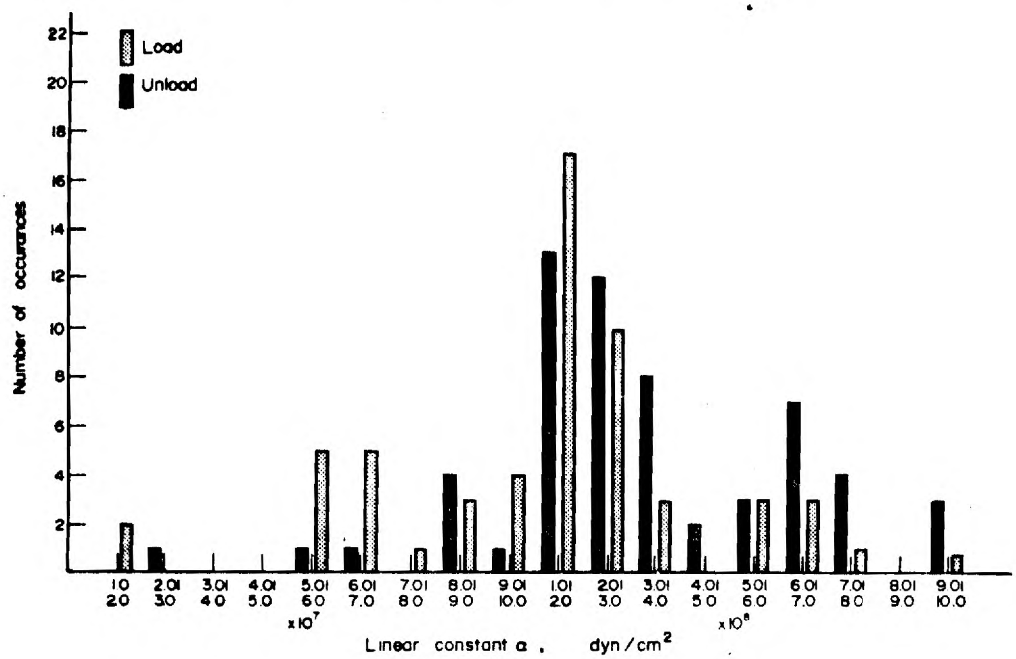

Fig. 2. Distribution of results for material parameter α.

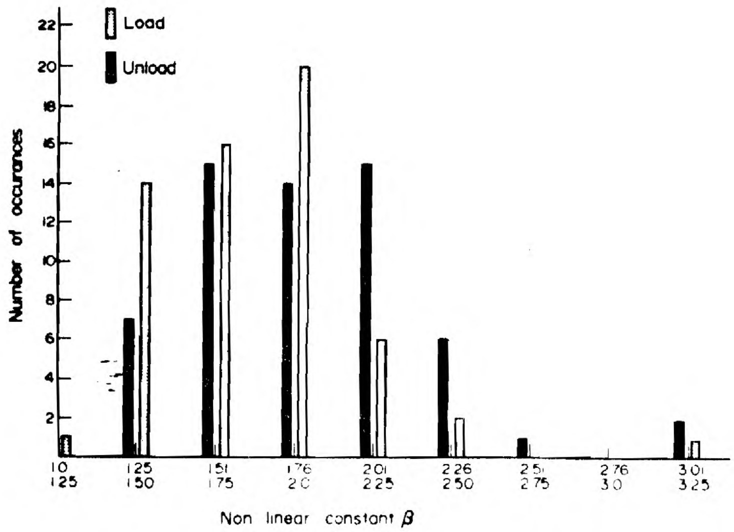

Fig. 3. Distribution of results for material parameter β.

Table 1. Summary of results of seventeen experiments

	Mean	S.D.
β (loading)	2.517	0.335
β (unloading)	3.491	0.324
α (loading)	$1.705 \times 10^8 \dfrac{\text{dyn}}{\text{cm}^2}$	0.052×10^8
α (unloading)	$1.928 \times 10^8 \dfrac{\text{dyn}}{\text{cm}^2}$	0.047×10^8
RMS error (% of max stress)	2.602%	0.088%

Consider the deformation which takes a point with spherical co-ordinates (R, θ, ϕ) in the undeformed geometry to a point (r, θ, ϕ) in the deformed geometry. This deformation field is analyzed in Green and Adkins (1960). An outline of their solution, as it applies here, follows.

From incompressibility, one obtains the equation for $r(R)$

$$\frac{\mathrm{d}r}{\mathrm{d}R} = \frac{R^2}{r^2}. \qquad (4)$$

Integrating gives

$$r^3 = R^3 - R_{HO}^3 + r_{HO}^3 \qquad (5)$$

for the heart and

$$r^3 = R^3 - R_{PI}^3 + r_{PI}^3 \qquad (6)$$

for the pericardium. Here R_{HO} and r_{HO}, are, respectively, the undeformed and deformed external radii for the heart, and R_{PI} and r_{PI} are, respectively, the undeformed and deformed internal radii for the pericardium.

For convenience, introduce the nondimensional co-ordinate $x \equiv r/R$. The strain invariants are then

$$I_1 = x^4 + \frac{2}{x^2} \qquad (7)$$
$$I_2 = \frac{1}{x^4} + 2x^2.$$

The stresses become

$$\tau^{rr} = 2x^2\Psi + p + x^4\Phi \qquad (8)$$
$$r^2\tau^{\theta\theta} = r^2\tau^{\phi\phi}\sin^2\theta = \frac{\Phi}{x^2} + \left(\frac{1}{x^4} + x^2\right)\Psi + p,$$

where p is an unknown scalar function of position.

The only non-trivial equilibrium equation may be given as

$$\frac{\mathrm{d}\tau^{rr}}{\mathrm{d}x} = 2(1 + x^3)\Phi + 2\left(x + \frac{1}{x^2}\right)\Psi. \qquad (9)$$

Finally, the boundary conditions are

$$\tau^{rr}(r_{HO}) = -P_{PF}$$
$$\tau^{rr}(r_{HI}) = -P_H \qquad ($$

for the heart and

$$\tau^{rr}(r_{PO}) = 0$$
$$\tau^{rr}(r_{PI}) = -P_{PF} \qquad ($$

for the pericardium. Here r_{HI} is the deformed intern radius of the heart, r_{PO} is the deformed external radi of the pericardium, P_H is the pressure in the heart a P_{PF} is the pressure in the pericardial fluid.

Equation (9) may be integrated for the heart a pericardium. Applying the boundary conditions, equ tions (10) and (11), to the results gives

$$P_H - P_{PF} = \int_{x_{HI}}^{x_{HO}} \left[2(1 + x^3)\Phi_H + 2\left(x + \frac{1}{x^2}\right)\Psi_H\right]\mathrm{d}x \qquad (1$$

$$P_{PF} = \int_{x_{PI}}^{x_{PO}} \left[2(1 + x^3)\Phi_P + 2\left(x + \frac{1}{x^2}\right)\Psi_P\right]\mathrm{d}x, \qquad (1$$

where Φ_P and Ψ_P follow from the elastic potential W for the pericardium and Φ_H and Ψ_H follow from t elastic potential W_H for the heart:

$$\Phi_P = 2\frac{\partial W_P}{\partial I_1} \qquad \Psi_P = 2\frac{\partial W_P}{\partial I_2}$$
$$\Phi_H = 2\frac{\partial W_H}{\partial I_1} \qquad \Psi_H = 2\frac{\partial W_P}{\partial I_2}.$$

In addition,

$$x_{HO} = \frac{r_{HO}}{R_{HO}} \qquad x_{HI} = \frac{r_{HI}}{R_{HI}}$$
$$x_{PO} = \frac{r_{PO}}{R_{PO}} \qquad x_{PI} = \frac{r_{PI}}{R_{PI}}.$$

Assuming the pericardial fluid to be incompressib and of volume V_{PF}, one obtains

$$V_{PF} = \frac{4}{3}\pi(R_{PI}^3 - R_{HO}^3) = \frac{4}{3}\pi(r_{PI}^3 - r_{HO}^3). \qquad (1$$

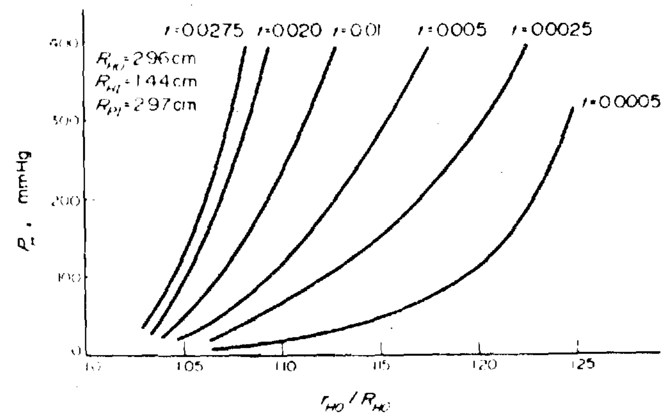

Fig. 4. The effect of varying pericardial thickness (pericarditis) on the internal pressure of the deformed heart.

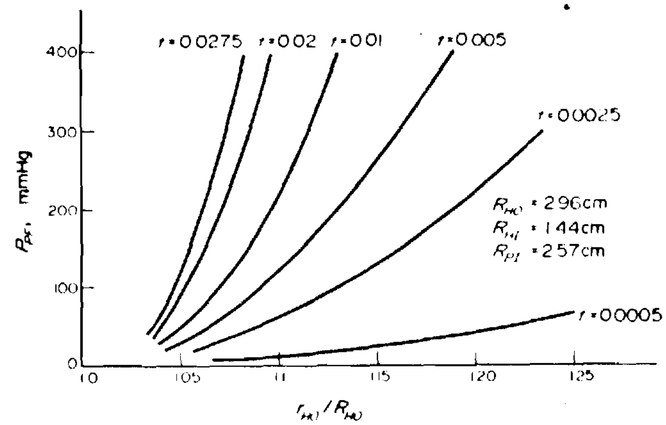

Fig. 5. The effect of varying pericardial thickness (pericarditis) on the pericardial fluid pressure of the deformed heart.

Given the initial dimensions, equations (5) and (6) may be used to calculate x_{HI} and x_{PO}, while equation (14) determines x_{PI}. With these, equations (12) and (13) determine P_H and P_{PF} for a deformation specified by x_{HO}.

RESULTS

Both Demiray (1976) and Mirsky (1973) proposed forms for W_H which are in good agreement with the experimental data of Sponitz *et al.* (1966). Following Demiray (1976), we set

$$W_H = \frac{\alpha_H}{2\beta_H}\left[e^{\beta_H(I_1 - 3)} - 1\right] \tag{15}$$

with $\beta_H = 1.256$ and $\alpha_H = 4550$ dyn/cm^2.

The pericardium was modeled using

$$W_P = \alpha_P(I_1 - 3)^{\beta_P} \tag{16}$$

with $\alpha_P = 1.816 \times 10^8$ dyn/cm^2 and $\beta_P = 3.004$, being the average of the values found in Table 1.

Normal pericardial fluid volumes for the dog range from 0.5 to 2.5 cm^3 (Holt, 1970). For simplicity, this volume is assumed to occupy the entire intrapericardial space.

Figures 4 and 5 illustrate the effect of simulated pericarditis on the pressure in the heart P_H and in the pericardial fluid P_{PF}. A pericardial fluid volume of 1.1 cm^3 was assumed. By way of comparison, the mean thickness of the specimens was 0.0233 cm.

CONCLUSION

The model developed shows the effects of pericardial thickness changes on pressures in the heart to be significant. Other pathological situations, such as pericardial effusion, could be expected to have similar effects. The further consideration of this problem, as well as the development of more complex models, such as those based on finite elements, or including viscous effects, awaits the results of bi-axial experiments now in progress.

Acknowledgements – I'd like to thank three Georgia Tech undergraduates for their help in this work. They are John Kennedy, who helped with the experiments and John Fay and William Youtie, who helped with some of the numerical work.

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To appear in Proceedings of the 10th Southeastern Conference on Theoretical and Applied Mechanics.

The Mechanics of the Pericardium: Pericardial Effusion*

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September 14, 1979

ABSTRACT

The influence of the pericardium on a model of cardiac function is studied for the case of simulated pericardial effusion. The model makes use of experimental data on the mechanical properties of the pericardium previously reported by one of us (RPV).

Both heart and pericardium are considered to be concentric elastic spheres separated by an incompressible fluid (pericardial fluid).

Introduction

The pericardium is the thin fibrous sac surrounding the heart of most vertebrates, including man. It serves to isolate and protect the heart from other thoracic structures and to help prevent cardiac hypertrophy. The pericardial sac also contains pericardial fluid which lubricates the heart.

Histologically, the pericardium consists of a thin membrane (visceral pericardium) surrounded by a relatively thick fibrous sac (parietal pericardium). The later is composed of elastin and collagen fibers; relative composition being a function of age in man (Holt 1970). It is these fibers which account for the mechanical strength of the tissue.

The pericardium is not essential to life and its removal does not result in any obvious disability. However, its restriction of free myocardial expansion can have life threatening consequences in cases of pericardial effusion (the infusion of fluid into the pericardial sac) and pericarditis (thickening of the pericardium) (Fowler 1970).

Elevated ventricular wall stresses may also result in ischemia of the ventricular wall (Mirsky 1973). This may be important in cases of pericarditis and pericardial effusion associated with myocardial infarction.

Much interest in biomechanics focuses on the development of mechanical models of the heart as a means of obtaining additional information from clinical measurements of cardiac function (e.g. Demiray 1976, Mirsky 1973, Voukydis 1972, a, b, c, Janz 1972, Gould et al. 1972, Ghista 1969 a, b, Mirsky, 1969, Wong et al. 1968). None of these models includes the pericardium.

In light of the above comments, it appears that the pericardium may be important in cardiac modelling. Of fundamental importance are the mechanical properties of the pericardium. Recently, Vito (1979) presented the results of a series of one-dimensional stretching experiments using dog pericardium. This paper uses these results, in a simplified model, to study the influence of the pericardium on the heart in the case of pericardial effusion.

Experiment

The experimental results will be briefly reviewed. The reader is referred to Vito (1979) for details.

A total of twenty experiments were conducted using tissue from ten dogs. Though specimens exhibited viscoelasticity under cyclic loading, individual load/unload cycles were modeled assuming elastic behavior.

Specimens were assumed to be homogeneous, isotropic and incompressible. Justifications for these may be found in Vito (1979).

Given these assumptions, the one dimensional stress-ellongation relationship for a prismatic bar is given by (Green et al. 1960)

$$T(\Lambda) = \left(\Lambda - \frac{1}{\Lambda^2}\right)\Phi_P + \left(1 - \frac{1}{\Lambda^3}\right)\Psi_P \tag{1}$$

where

$$\Lambda = \frac{L}{L_o} \qquad \Phi_P = 2\,\frac{\partial W_P}{\partial I_1} \qquad \Psi_P = 2\,\frac{\partial W_P}{\partial I_2} \tag{2}$$

Here T is the stress referenced to the underformed cross sectional area, L is the deformed and L_o the undeformed length, I_1 and I_2 are strain invariants and $W_P = W_P\,(I_1\,I_2)$ is the strain energy density function for the pericardium.

The function W_P is not uniquely determined by one-dimensional tests. However, any choice for W_P must agree with all the known data.

Several possible functional forms for W_P may be found in the literature on soft tissues (e.g. Demeray and Vito 1976, Fung et al. 1979).

Choosing

$$W_P = \alpha_P \left(I_1 - 3\right)^{\beta_P} \tag{3}$$

resulted in a good fit, in a least squares sense, to the data. Note that equation (3) is a generalization of the familiar Mooney characterization of rubber elasticity.

Table (1) summarizes the experimental results.

Model

The heart will be modeled as an elastic sphere composed of homogeneous, isotropic incompressible material. This model was considered by Mirsky (1973), Demiray (1976) and others. The pericardium, not previously considered, will be assumed to be a second elastic sphere, similarly composed and concentric with the first.

Consider the deformation which takes a point with spherical coordinates (R, θ, ϕ) in the undeformed geometry to a point (r, θ, ϕ) in the deformed geometry. This deformation field is analyzed in Green et (1960). An outline of their solution, appropriately modified, follows.

From incompressibility, one obtains the equation for r (R):

$$\frac{dr}{dR} = \frac{R^2}{r^2}$$

Integrating gives

$$r^3 = R^3 - r_{HO}^{\ 3} + r_{HO}^{\ 3} \tag{4}$$

for the heart and

$$r^3 = R^3 - R_{PI}^{\ 3} + r_{PI}^{\ 3} \tag{5}$$

for the pericardium. Here R_{HO} and r_{HO} are, respectively, the undeformed and deformed external radii for the heart and R_{PI} and r_{PI} are, respectively, the undeformed and deformed internal radii for the pericardium.

For convenience, introduce the nondimensional co-ordinate $x \equiv r/R$. The strain invariants are then

$$I_1 = x^4 + \frac{2}{x^2}$$
$$I_2 = \frac{1}{x^4} + 2x^2 \tag{6}$$

The stresses become

$$\tau^{rr} = 2x^2\Psi + x^4\,\Phi + p$$
$$r^2\tau^{\theta\theta} = r^2\tau^{\phi\phi}\sin^2\theta = \frac{\Phi}{x^2} + \left(\frac{1}{x^4} + x^2\right)\Psi + p \tag{7}$$

where p is an unknown scalar function of position and Φ and Ψ are determined from the strain energy density function.

The only non-trivial equilibrium equation is

$$\frac{d\tau^{rr}}{dx} = 2\left(1 + x^3\right)\Phi + 2\left(x + \frac{1}{x^2}\right)\Psi \tag{8}$$

The boundary conditions are

$$\tau^{rr}\left(r_{HO}\right) = -P_{PF}$$
$$\tau^{rr}\left(r_{HI}\right) = -P_H \tag{9}$$

for the heart and

$$\tau^{rr}\left(r_{PO}\right) = 0$$
$$\tau^{rr}\left(r_{PI}\right) = P_{PF} \tag{10}$$

for the pericardium. Here r_{HI} is the deformed internal radius of the heart, r_{PO} is the deformed external radius of the pericardium, P_H is the pressure in the heart and P_{PF} is the pressure in the pericardial fluid.

Equation (8) may be integrated for the heart and pericardium. Applying the boundary conditions (9) and (10) to the results gives

$$P_H - P_{PF} = \int_{x_{HI}}^{x_{HO}} 2\left\{\left(1 + x^3\right)\Phi_H + \left(x + \frac{1}{x^2}\right)\Psi_H\right\} dx$$

$$P_{PF} = \int_{x_{PI}}^{x_{PO}} 2\left\{\left(1 + x^3\right)\Phi_P + \left(x + \frac{1}{x^2}\right)\Psi\right\} dx \qquad (11)$$

where Φ_p and Ψ follow form the elastic potential W_P for the pericardium and Φ_H and Ψ_H follow from the elastic potential W_H for the heart:

$$\Phi_p = 2\frac{\partial W_P}{\partial I_1} \qquad \Psi_P = 2\frac{\partial W_P}{\partial I_2}$$

$$\Phi_H = 2\frac{\partial W_H}{\partial I_1} \qquad \Psi_H = 2\frac{\partial W_H}{\partial I_2} \qquad (12)$$

In addition,

$$x_{HO} = \frac{r_{HO}}{R_{HO}} \qquad x_{HI} = \frac{r_{HI}}{R_{HI}}$$

$$x_{PO} = \frac{r_{PO}}{R_{PO}} \qquad x_{PI} = \frac{r_{PI}}{R_{PI}}$$

Assuming the pericardial fluid to be incompressible and of volume V_{PF}, one obtains

$$V_{PF} = \frac{4}{3}\pi\left(R_{PI}^3 - R_{HO}^3\right) = \frac{4}{3}\pi\left(r_{PI}^3 - r_{HO}^3\right) \qquad (13)$$

Given the initial dimensions, equations (4), (5), (11) and (13) may be combined to give three equations relating P_{PF}, P_H, and x_{HO}.

Results

Both Demiray (1976) and Mirsky (1973) proposed forms for W_H which are in good agreement with the experimental data of Sponitz et al (1966). Following Demiray (1976), we set

$$W_H = \frac{\alpha_H}{\beta_H}\left[e^{\beta_H (I_1 - 3)} - 1\right] \tag{14}$$

with $\beta_H = 1.256$ and $\alpha_H = 4550$ dynes/cm^2.

The pericardium is modeled using equation (3) with $\alpha_p = 1.816 \times 10^8$ dynes/cm^2 and $\beta_p = 3.004$ being the average valves found in Table (1).

Normal pericardial fluid volumes for the dog range from 0.5 to 2.5 cc. A normal pericardial fluid volume of 1.5 cc was assumed.

Figures (1) and (2) illustrate the effect of similated pericardial effusion on P_H and P_{PF} for various increases in pericardial fluid volume of ΔV_{PF} (in cc).

Discussion

For the assumptions stated, one-dimensional data is not sufficient for the unique determination of W. Such a determination requires a bi-axial experiment in which the principal strains can be independently varied. These experiments have, to the author's knowledge, not been done.

Though our model is a simple one, it shows the importance of the pericardium in cardiac modelling. Figures (1) and (2) show the effects of increases in pericardial fluid volume to be significant. Pericardial fluid pressure is increased. Similar increases in the stresses in the heart wall can be expected.

Consideration of more complex models, such as those based on the finite element technique, await further experimental data.

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Table 1: Summary of results for seventeen one dimensional **experiments.**

	MEAN	S. D.
β (loading)	2.517	0.335
β (unloading)	3.491	0.324
α (loading)	$1.705 \times 10^{8} \frac{dynes}{cm^2}$	0.052×10^{8}
α (unloading)	$1.928 \times 10^{8} \frac{dynes}{cm^2}$	0.047×10^{8}
RMS error (% of max stress)	2.602%	0.088%

Captions for Figures

Figure 1 The effect of pericardial fluid volume change ΔV_{PF} (cc) on P_H, the pressure in the heart.

Figure 2 The effect of pericardial fluid volume changes ΔV_{PF} (cc) on P_{PF}, the pericardial fluid pressure.

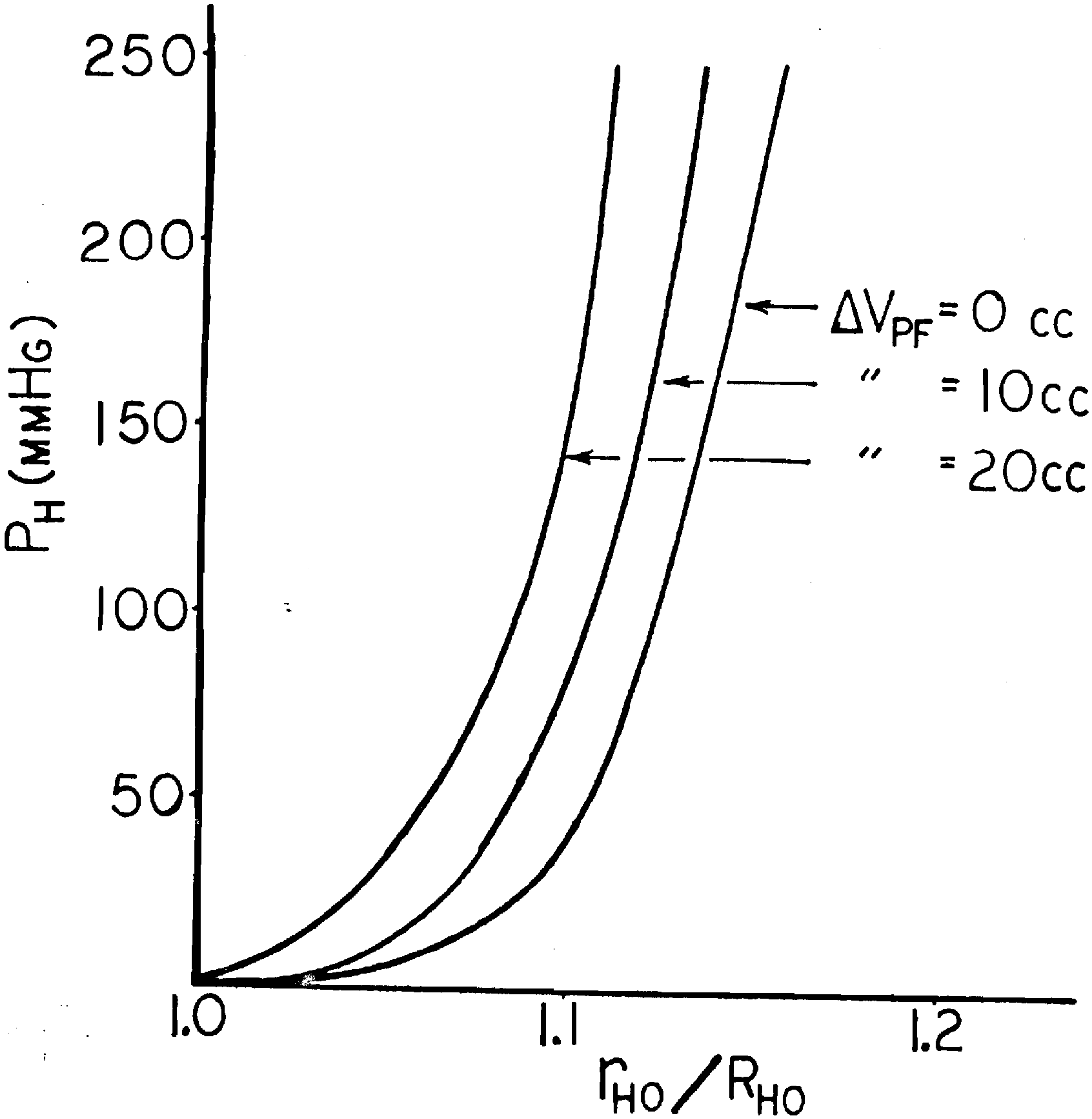
250
200
150
100
50
1.0
1.1
1.2
P_H (MMHG)
r_{HO}/R_{HO}
ΔV_{PF} = 0 cc
" = 10cc
" = 20cc

Fig 1

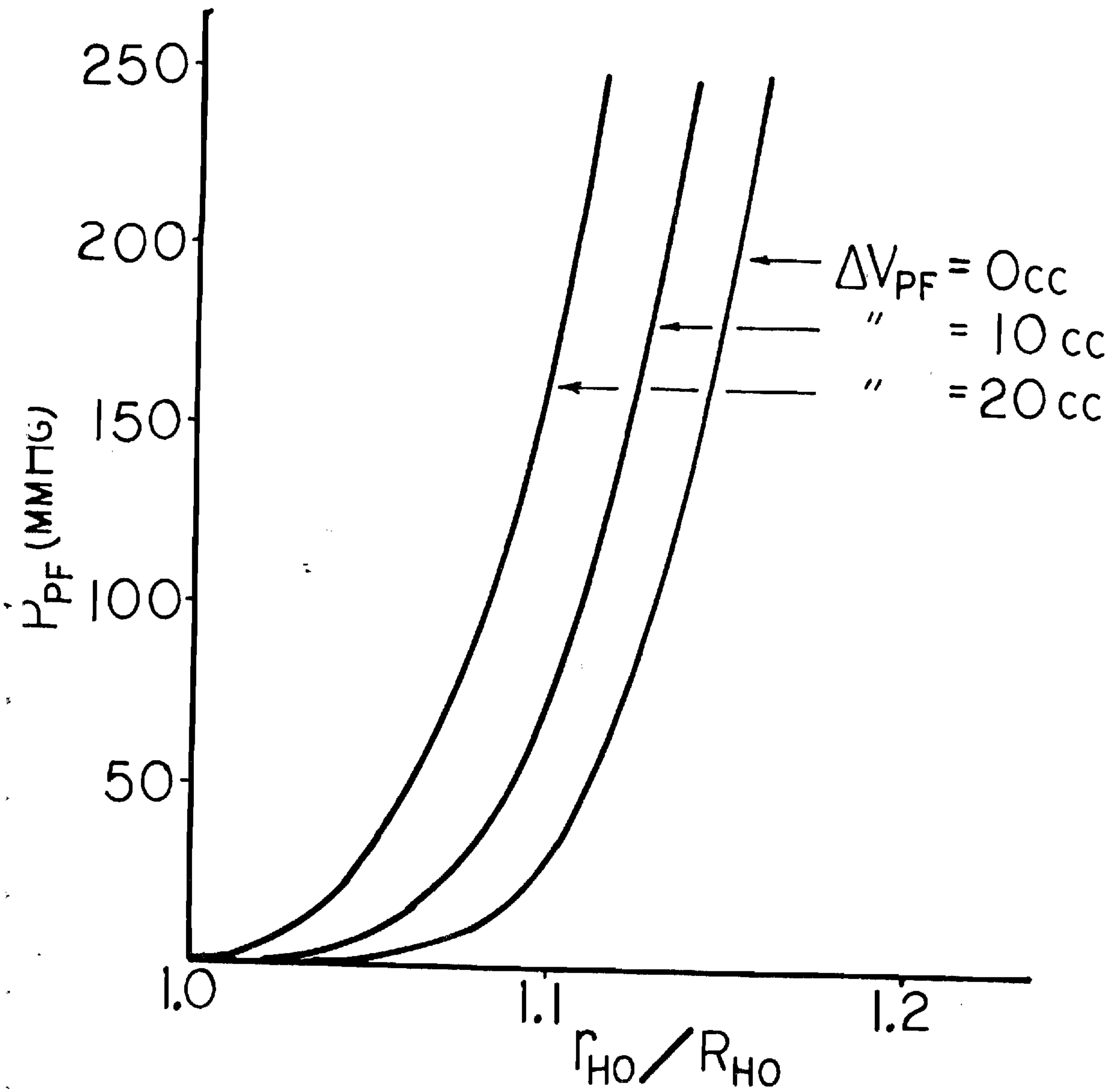

FIG 2